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Techniques in Vascular and Interventional Radiology

Evaluation and Initial Stabilization of Undifferentiated Shock

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Vascular Interventional Radiologists (VIRs) are increasingly involved in consultation and management of patients with circulatory shock. The ability to recognize the underlying pathophysiology of a patient's shock and treat appropriately is therefore critical to VIR practice. Here, we discuss the initial evaluation and stabilization of undifferentiated shock. Shock is defined as tissue hypoperfusion from inadequate oxygen delivery and/or impaired tissue oxygen utilization. The etiologies of shock can be grouped into 3 main categories: distributive, hypovolemic, cardiogenic, and obstructive; each of which are associated with characteristic hemodynamic and metabolic derangements such as decreased preload or impaired cardiac contractility. Presentation typically involves 1) hypotension and tachycardia as indicators of impaired cardiac output, and 2) evidence of ongoing end-organ hypoperfusion such as oliguria, altered mental status, and lactic acidosis. Initial management includes control of airway and oxygenation/ventilation, identification of underlying hemodynamic and cardiovascular derangements, and support of cardiac output with intravenous fluid (IVF), blood products, diuresis, and/or vasopressors and inotropes. More detailed hemodynamic analysis can be performed with arterial catheters, blood gases, pulmonary artery catheters, and echocardiography to further tailor therapy. Finally, definitive evaluation for causative etiology should be initiated immediately with biochemical studies, cultures, imaging, and hemodynamic assessment so that etiology-specific treatment (e.g. antibiotics, blood products) can be initiated.

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Introduction

Shock is a life-threatening condition characterized by tissue hypoperfusion leading to cellular and organ dysfunction.[1] It can be caused by a wide spectrum of pathologies that impair oxygen delivery to tissues and/or impair their ability to utilize delivered oxygen. Vascular Interventional Radiologists (VIRs) must be prepared to manage shock, especially when complications arise during procedures or in consultation or management of critically-ill patients—frequently encountered examples include hemorrhagic shock after trauma, obstructive shock from massive pulmonary embolism (PE), and septic shock from obstructive uropathy. Undifferentiated shock, where the cause is unclear at presentation, requires a systematic approach to identify the underlying pathophysiology while initiating prompt stabilization measures. Key components of diagnosis and treatment include recognition of shock through physical examination, imaging, and laboratory evaluation; securing airway, breathing, and circulation ("ABCs"); hemodynamic stabilization and assessment; correction of underlying etiologies; and restoration of oxygen delivery. This article aims to provide a concise guide for IRs on the presentation and pathophysiology of shock, evaluation and initial stabilization of undifferentiated shock, methods for diagnosing underlying etiologies, identification of hemodynamic derangements, and performance of hemodynamically guided resuscitation to improve patient outcomes.

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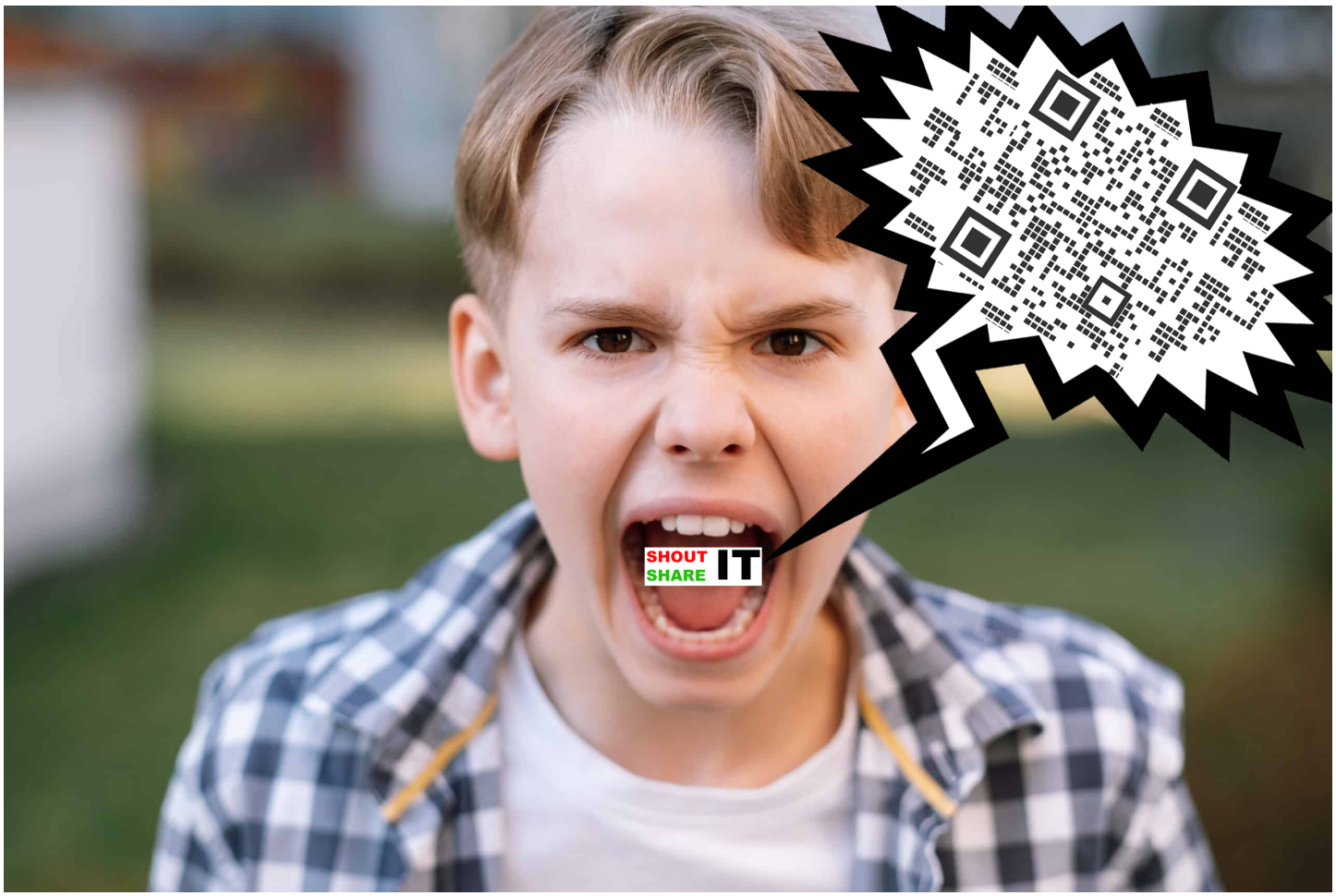
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Definition and Presentation

Circulatory shock can be defined as a state where tissue oxygenation is inadequate to meet the demands of aerobic metabolism (i.e., a state of hypoxia), leading to cellular and organ dysfunction.[2] Shock presents with signs and symptoms of end-organ hypoperfusion that signal ongoing organ dysfunction. These include altered mental status, lactic acid accumulation from anaerobic metabolism and resulting anion gap acidosis, oliguria (urine output < 0.5 mL/kg/hr), acute liver failure ("shock liver"), and skin mottling/clamminess. Notably, hypotension is often associated with shock, and sustained hypotension can meaningfully reduce oxygen delivery to tissues, but the presence of hypotension does not necessarily indicate ongoing end-organ dysfunction from tissue hypoxia. Furthermore, not all patients in shock will be hypotensive, and some patients who are in the early phases of shock may even be normotensive or hypertensive, depending on the physiologic compensatory reserve and medical history of the particular patient. For example, a relative decrease in MAP > 40 mmHg should be considered hypotension in patients with chronic hypertension.[3] Therefore, it is important to recognize hypotension as a warning sign associated with shock in the context of other clinical, biochemical, and hemodynamic features.

Pathophysiology

Shock typically occurs due to some combination of decreased oxygen delivery to tissues, impaired oxygen utilization by tissues, and/or elevated tissue oxygen demands. If prolonged and severe, the oxygen available to tissues for uptake will be insufficient to support aerobic metabolism, and anaerobic metabolism will occur, followed by organ dysfunction. Shock can be precipitated by any pathology or condition that decreases oxygen delivery or impairs oxygen utilization. Oxygen delivery to tissues (DO_2) is reliant on cardiac output (CO); therefore, any process that decreases CO can lead to tissue hypoxia. CO in turn is determined by 3 major hemodynamic variables: heart rate, preload (the volume of blood return to the right heart), afterload (the resistance against which the heart must pump to eject blood), and contractility (the contractile strength of the left ventricle in ejecting blood). The various etiologies of shock can be divided into 3 broad categories, distributive (66%) (e.g. septic shock, anaphylactic shock, neurogenic shock), hypovolemic (16%) (e.g. hemorrhagic, other fluid losses), cardiogenic (16%) (e.g. acute myocardial infarction, arrhythmias), and obstructive (2%) (e.g. tension pneumothorax, PE, pericardial tamponade), grouped according to the unique pathophysiologic mechanisms by which they negatively impact CO (Table 1).[1,4,5]

- Distributive shock is characterized by pathophysiologic peripheral vasodilation resulting in decreased systemic vascular resistance (SVR), effectively leading to "pooling" of blood volume in the periphery and reduced preload. Decreased SVR also causes hypotension (mean arterial pressure = CO × SVR). Etiologies include sepsis, spinal cord injury, and anaphylaxis:
  - Septic shock: Vasoactive toxins and cytokines induce pathologic peripheral vasodilation, prompting initial compensatory increase in HR. In addition to decreased oxygen delivery from reduced preload, cellular oxygen utilization is also impaired due to mitochondrial dysfunction, a phenomenon known as "cytopathic hypoxia." As septic shock progresses into later stages, cardiac function deteriorates, leading to decreased CO.[6]
  - Neurogenic shock: Loss of sympathetic tone, typically due to a high spinal cord injury (SCI) above T6 spinal level, causes loss of peripheral vascular tone and decreased SVR.
  - Anaphylactic shock: Release of vasoactive mediators such as histamine leads to rapid peripheral vasodilation and decreased SVR.
- Hypovolemic shock, whether from hemorrhagic or nonhemorrhagic intravascular volume loss, results from a decrease in preload that is significant enough to decrease CO. Initial phases usually feature compensatory increases in HR and SVR in an attempt to maintain

Table 1 Clinical and Hemodynamic Parameters in Different Types of Shock

Type	Skin Temperature	JVP	Passive Leg Raise	CVP	PCWP	HR	CO	SVR	SvO_2
Distributive		↓	↑PP						
Septic (Early)	Warm			↓	↓	↑	↑	↓*	↑
Septic (Late)	Warm/Cool			↓	↓	↑	↓	↓*	↑
Neurogenic	Warm			↓	↓	↓	↓	↓*	↓
Anaphylactic	Warm			↓	↓	↑	↓	↓*	↑
Hypovolemic	Cool	↓	↑PP	↓*	↓*	↑	↓	↑	↓
Cardiogenic	Cool	↑	↔PP	↑	↑	↑/↓	↓*	↑	↓
Obstructive	Cool	↑	↔PP						
Pulmonary (PE, severe PHTN)				↑	↓	↑	↓*	↑	↓
Tamponade (pericardial tamponade, tension PTX, ACS)		↑		↑	↑	↑	↓*	↑	↓

Abbreviations: ACS, abdominal compartment syndrome; CO, cardiac output; CVP, central venous pressure; HR, heart rate; JVP, jugular venous pressure; PCWP, pulmonary capillary wedge pressure; PE, pulmonary embolism; PHTN, pulmonary hypertension; PP, pulse pressure; PTX, pneumothorax; SvO_2, mixed venous oxygen saturation; SVR, systemic vascular resistance.

*Primary driver of shock.

mean arterial pressure (MAP), with decompensation leading to significant hypotension.

- Cardiogenic shock occurs due to direct decrease in CO from causes such as acute myocardial infarction, arrhythmias, or cardiomyopathies.
- Obstructive shock can be subdivided into etiologies stemming from increased pulmonary vascular resistance and those stemming from extrinsic mechanical compression:
  - Pulmonary vascular causes: Massive PE or severe pulmonary hypertension result in significantly increased right ventricular (RV) afterload, leading to elevated right-sided chamber pressures and RV distension with bowing of the interventricular septum into the left ventricle (LV). This ultimately impedes LV filling, resulting in decreased CO.
  - Extrinsic mechanical compression: Pericardial effusion and tension pneumothorax exert extrinsic pressure on all cardiac chambers, effectively raising biventricular afterload. This prevents diastolic filling of first the right heart, then left heart, eventually resulting in decreased CO. Abdominal compartment syndrome compresses the IVC and impairs venous return to the right heart, leading to decreased LV filling and decreased CO.

Initial Stabilization and Evaluation

Clinical management of the shock patient can be divided into 3 functional goals: emergent stabilization, diagnostic workup to assess etiology and category of shock, detailed assessment of hemodynamic derangements, and resuscitation.

Emergent stabilization, particularly of the patient presenting *in extremis*, should start with evaluation of the airway, breathing, and circulation ("ABCs") along with continuous monitoring of vital signs.[7] Recognizing the need to call for help, such as activating a rapid response or code team, is of utmost importance, particularly in the procedure suite where limited staffing can pose challenges. Respiratory oxygenation and ventilation should be optimized to counteract the reduced oxygen delivery and increased tissue oxygen demands induced by shock states. This may involve the use of supplemental oxygen delivery, noninvasive positive pressure ventilation, or endotracheal intubation and mechanical ventilation. Venous access should be achieved with multiple large-bore IVs and/or a central venous catheter (CVC). An arterial catheter should be inserted (typically in the radial artery) for accurate real-time blood pressure monitoring and procurement of arterial blood gas and lab samples. Severity of shock can be assessed by inserting a Foley catheter and measuring hourly urine output, as well as assessing serum lactate at regular intervals. Hypotension should be corrected with intravenous administration of balanced crystalloid fluids (e.g. lactated ringer's, normal saline) in undifferentiated shock or blood products in hemorrhagic shock. Any arrhythmia causing hypotension must also be treated with ACLS protocol.

After initial stabilization, focus should be turned towards determining the etiology and category of a patient's shock in order to facilitate definitive treatment. In some patients, the etiology may be strongly suggested by the recent clinical course. For example, a patient recovering from a percutaneous nephrostomy to treat obstructive pyonephrosis is likely septic, a patient undergoing iliocaval thrombolysis may have developed a massive PE, and a patient arriving hypotensive with a gunshot wound is likely hemorrhaging.[8] Initial diagnostic workup should flow from the history and recent clinical course, and etiology-specific monitoring and treatment should be initiated immediately if a diagnosis is strongly suspected.

A basic initial workup for shock includes twelve-lead ECG, CMP, CBC with differential, lactate, respiratory/urine/blood cultures, chest radiograph, focused cardiac ultrasound (FoCUS), and additional point of care ultrasonography to assess for free fluid and hemorrhage. Notably, FoCUS is a powerful bedside tool that can rapidly identify pathology and patterns of cardiac injury that can impact cardiac function and precipitate shock, such as cardiac tamponade from an effusion, right ventricular failure from massive PE, or acute valvular regurgitation. Additionally, it can be used for hemodynamic assessment by estimating CO and intravascular fluid status.[9] In addition to resuscitation to restore CO, several etiologies of shock require emergent disease-specific workup and intervention:

1. Sepsis: initial complete blood count (CBC) with differential and respiratory, urine, and blood cultures (x2 bottles) should be obtained; crystalloid IV fluids 30 ml/kg should be administered within the first hour to maintain CO; and broad-spectrum IV antibiotics should be initiated ideally after cultures are obtained.[10] Examination/imaging for infectious sources such as abscesses, vegetations, wounds, surgical implants or devices is necessary to achieve source control.
2. Adrenal insufficiency/thyroid storm: hydrocortisone bolus for adrenal crisis and β-blockers, thionamide, iodine, propylthiouracil, and steroids for thyroid storm.[11,12]
3. Anaphylaxis: requires immediate intramuscular epinephrine injection, and can be additionally managed with oxygen, antihistamines, steroids, and β-agonists.[13]
4. Hemorrhage: restoring blood volume and correcting coagulopathy is of utmost importance, using either whole blood or a 1:1:1 ratio of component therapy (pRBC:platelets:FFP). If further coagulopathy is evident after initial resuscitation, thromboelastography (TEG) or rotational thromboelastometry (ROTEM) may be used for more directed component resuscitation.[14] Ultimately, the source of bleeding must be identified and controlled, either by directed pressure, angioembolization, or surgery. Baseline studies should include hemoglobin (Hb) and coagulation profile (PT/PTT/INR/fibrinogen).
5. Myocardial infarction (MI): troponin and 12-lead ECG should be obtained emergently along with FoCUS/

echocardiography if cardiogenic shock from MI is suspected. Arrangements for catheterization lab transfer and/or IV thrombolysis should be initiated.
6. Decompensated heart failure: this can quickly lead to pulmonary edema and respiratory failure in addition to shock; therefore, diuresis and supplemental oxygenation should be administered where appropriate. Chest radiograph, BNP, and troponin values should be obtained and trended if suspected.
7. Massive PE: can quickly lead to right ventricular failure and cardiopulmonary collapse. FoCUS/echocardiography and CT pulmonary angiography should be performed urgently to characterize hemodynamic status and thrombus size, which will determine definitive treatment with IV thrombolysis, catheter-directed thrombolysis/thrombectomy, and/or surgical embolectomy.[15]
8. Tension pneumothorax: needle thoracostomy must be performed to relieve pressure on the lungs, thereby restoring venous return and improving shock. Follow-up placement of a chest tube is often required to prevent re-accumulation of air.[16] Urgent chest radiography or CT should be performed but should not delay intervention.
9. Pericardial tamponade: emergent diagnosis with FoCUS/echocardiography and ultrasound-guided pericardiocentesis and drain placement is mandatory in unstable patients.

Detailed Hemodynamic Assessment

As previously discussed, each category of shock is associated with characteristic hemodynamic changes (Table 1). Therefore, if shock state persists after initial stabilization and treatment of readily identifiable etiologies, a detailed hemodynamic assessment should be performed to identify the hemodynamic category of shock and subsequently tailor resuscitation to particular hemodynamic derangements. This broadly involves assessing CO (including its determinants — preload, afterload, and myocardial contractility), intravascular fluid status and fluid responsiveness, and SVR.

Hemodynamic Assessment Tools

Several tools are available to assess these variables, including the pulmonary artery catheter (PAC), CVC, arterial pressure waveform analysis (APWA), and FoCUS. The PAC has long been the gold standard to quantitatively measure hemodynamic parameters and monitor their trajectory in real time. While it is the most invasive option with associated risks including arrhythmia and pulmonary artery rupture, it is also the most accurate, comprehensive, and reliable assessment available. APWA has been billed as a minimally-invasive replacement for PAC — it involves interpretation of data from a sensor such as FloTrac (Edwards Lifesciences, Irvine CA) that is attached to a standard arterial pressure catheter. APWA measurements may not be as accurate as PAC measurements, particularly in cases of low SVR, pressure lability and use of vasopressors, arrhythmia or ectopy, poor arterial pressure waveform quality, and spontaneous respiration.[17] FoCUS is a useful bedside noninvasive adjunct that can, in addition to identifying anatomic pathology and patterns of cardiac injury, quantitatively estimate certain hemodynamic parameters.

Intravascular Volume Status

Typically, the first step in detailed hemodynamic assessment is determining whether the patient has depleted intravascular volume (hypovolemia) and may benefit from fluid administration or is fluid overloaded (hypervolemic) and would benefit from diuresis. Both states may impair CO — hypovolemia decreases CO by reducing cardiac preload, while hypervolemia can 1) lead to venous congestion and decreased organ perfusion, and 2) overwhelm the Frank-Starling mechanism of the heart leading to decreased myocardial contractility and CO. While there is no assay to directly measure intravascular volume, data points from focused physical examination, imaging, and invasive hemodynamic monitoring can be combined to provide reasonable accuracy in suggesting either hypo- or hypervolemia.

On exam, hypovolemic patients may have dry mucus membranes and low urine output (< 400-500 ml per day or < 0.3-0.5 ml/kg/hour). Hypervolemic patients may have jugular venous distension, lung crackles or respiratory distress indicating pulmonary edema, and anasarca. Pulmonary edema may be confirmed on chest radiograph or CT.

Several ultrasound protocols exist to evaluate intravascular volume status. The intrahepatic IVC diameter and collapsibility with respiration can be measured — in the hypovolemic patient, respirophasic IVC collapse is accentuated, while hypervolemic patients may have plump and noncollapsible IVC.[18] FoCUS may show underfilled and collapsing chambers in hypovolemia versus distended chambers in hypervolemia.[19] The Venous Excess Ultrasound Score (VExUS) is a protocol that integrates pulsed-wave Doppler waveform analysis of the IVC and hepatic, portal, and renal veins into a score that indicates the severity of overall systemic venous congestion due to hypervolemia, if present.[20]

Invasive measurement of central venous pressure (CVP) with a CVC or PAC can be performed — under standard conditions, pressure is a function of chamber filling volumes. If this correlation is assumed, low CVP (< 5 mmHg) may indicate decreased preload/venous return to the right heart and hypovolemia, while elevated CVP (> 10 mmHg) may indicate volume overload. In practice, the trend of CVP and response to intravenous fluid administration or diuresis is more reliable an indicator of volume status than the absolute number.

Notably, there are several conditions that can confound interpretation of CVP and sonographic examination. Heart failure, cardiac tamponade, constrictive pericarditis, pulmonary hypertension, cardiomyopathies, and tricuspid regurgitation can all increase cardiac and IVC filling pressures independent of intravascular volume.[21] The IVC can also be

collapsed by increased intra-abdominal pressure or dilated by increased intrathoracic pressure.

Lastly, a patient's hemodynamic response to a fluid challenge can act as a diagnostic test to elucidate volume status. If CO improves with a fluid challenge (i.e. the patient is "fluid-responsive"), then the patient may be hypovolemic, while no change or worsening in hemodynamics may indicate euvolemia or hypervolemia. The fluid challenge can be performed either with 500 ml crystalloid (or 200 ml colloid) intravenous fluid bolus administration or passive leg raise (equivalent to IV fluid bolus of 150-750 ml). A positive response is defined by an increase in CO, which can be directly or indirectly assessed by the following methods:

1. Direct calculation of CO via PAC or APWA.
2. Decreased respirophasic variation in arterial pulse pressure and/or cardiac stroke volume (SV) in mechanically ventilated patients: these measurements are based on the principle that the respiratory cycle drives normal variation in cardiac SV, and therefore CO (negative intrathoracic pressure with inspiration → decreased LV preload → decreased SV → decreased CO). In hypovolemic patients, the drop in CO during inspiration is exaggerated due to low preload. This manifests as exaggerated variation in the arterial pulse pressure across the respiratory cycle as measured on an arterial line, and exaggerated stroke volume variation (SVV) as measured on APWA. In hypovolemic patients, a fluid challenge should decrease pulse pressure variation (PPV) and SVV. Notably, these measurements have only been validated with mechanical ventilation with 8 ml/kg tidal volumes in the absence of arrythmia, RV failure, or high respiratory rate.

Importantly, improvement in hypotension with fluid administration is often used as a quick indicator of hypovolemia; however, this should be interpreted with caution, as a change in blood pressure does not necessarily indicate an underlying change in CO.

Hemodynamic Parameters

Cardiac output (CO), which is commonly converted to cardiac index (CI) by dividing by body surface area, can be calculated with a PAC by either the Fick or thermodilution methods, and can also be estimated using APWA. Stroke volume (SV) can be calculated with FoCUS, which is then multiplied by heart rate to yield CO.

Chamber filling pressures can be measured to give an indication of chamber filling volumes (i.e. preload). The CVP measures right atrial pressure, which is equal to right ventricular end-diastolic (filling) pressure in the absence of tricuspid valve disease and can be measured from PAC or CVC as described previously. Left ventricular filling pressure is indirectly measured through the PAC positioned in a distal pulmonary artery with occlusion balloon inflated—in the absence of mitral valve disease, the pulmonary artery occlusion pressure (PAOP) equals the left atrial pressure, which in turn equals the left ventricular end-diastolic (filling) pressure.

Systemic vascular resistance (SVR) approximates the overall hydraulic resistance in the systemic vasculature and reflects the afterload that the left ventricle must pump against. SVR is calculated by subtracting CVP from MAP and dividing the result by CO, and therefore can be obtained using information from a PAC in conjunction with an arterial line, or from APWA with CVP entered manually.

While not a true hemodynamic variable, the oxygen saturation of venous blood serves as an indicator of adequate oxygen delivery to tissues. As overall oxygen delivery decreases in most shock states, tissues compensate to maintain oxygen utilization levels sufficient for supporting aerobic metabolism by extracting more oxygen from hemoglobin in arterioles and capillaries, resulting in decreased oxygen saturation of venous blood returning to the heart. Venous oxygen saturation is measured most accurately from a pulmonary artery blood sample obtained via PAC (termed "mixed venous oxygen saturation," or SvO2). If pulmonary arterial blood cannot be obtained, the oxygen saturation of central venous blood obtained via CVC (termed "central venous oxygen saturation", or ScvO2) can serve as an approximation. Normal SvO2 ranges from 60% to 80% (correlating with ScvO2 of 70%-80%). Assuming normal hemoglobin concentration and arterial saturation, the tissues can compensate for decreased oxygen delivery until SvO2 reaches ~50% (ScvO2 of ~70%), below which point no further oxygen can be extracted, aerobic metabolism cannot continue, and shock occurs.[22]

Hemodynamic Profiles of Shock

Patients with hypovolemic shock and those with distributive shock will typically show signs of intravascular fluid depletion and fluid responsiveness—the clinical history can usually determine which of the 2 categories a patient falls into. SVR is a classic differentiator: distributive shock is defined by processes that pathologically trigger peripheral vasodilation and decrease SVR, while in hypovolemic shock, the body mounts an adrenergic response to shunt blood from the extremities to the core by inducing peripheral vasoconstriction, which increases SVR. Early hypovolemic and distributive shock can be compensated by increasing heart rate and myocardial contractility through sympathetic tone. In distributive shock, this hyperdynamic circulation coupled with dysfunction in tissue oxygen utilization can lead to elevated SvO2/ScvO2 as tissues have less time to pull oxygen off hemoglobin as it speeds through the capillaries.

Cardiogenic shock is caused by processes intrinsic to the heart that impair its ability to generate sufficient output; therefore, CO and CI will be depressed. While decompensated shock from any category will eventually lead to cardiac dysfunction, hypokinesis, and decreased CO, cardiogenic shock is the only category that is initiated by this dysfunction. Poor forward flow also leads to elevated chamber filling pressures as blood volume "backs up," reflected in elevated CVP and/or PAOP. The body will mount an initial adrenergic

response, resulting in peripheral vasoconstriction and high SVR; however, with decompensation, this tone may be lost, and SVR may be low. SvO2 and ScvO2 are classically reduced <50% and <70%, respectively, as tissues compensate for poor overall oxygen delivery by extracting more oxygen from hemoglobin that is circulated.

Obstructive shock is defined by obstruction at some point in the cardiopulmonary circuit leading to impaired filling of the cardiac chambers and/or impaired output from right heart to left heart. It manifests with 1 of 2 classic hemodynamic profiles depending on the underlying etiology of obstruction. With massive PE or pulmonary hypertension, there is an acute increase in right ventricular afterload, leading to increased right heart pressures (CVP). The increased resistance to blood traveling from right to left heart also leads to decreased left-sided filling volumes, manifesting as normal or decreased PAOP. In contrast, with cardiac tamponade, extrinsic compression of all chambers of the heart also causes elevated CVP, but PAOP is elevated in this scenario due to the extrinsic compression despite decreased left-sided filling volumes.

Resuscitation

Once a patient's hemodynamic profile and category of shock has been characterized and etiology-specific treatments initiated, management of shock shifts to resuscitation, or targeted reversal of hemodynamic derangements. This is accomplished through optimization of fluid status using intravenous crystalloids/colloids or diuresis, and administration of vasopressors and/or inotropes to maximize preload, afterload, and myocardial contractility.

Pharmacology of Vasopressors and Inotropes

Vasopressors and inotropes are critical pharmacologic agents used to restore CO in shock. They accomplish this by acting on different receptors in the heart and/or vasculature to counteract certain hemodynamic derangements, namely peripheral vasodilation and impaired myocardial contractility, caused by different categories of shock.[23] Therefore, understanding the mechanism of action and hemodynamic effect of each agent is crucial to correcting the unique pathophysiology of each category of shock.

The main mechanism of action of vasopressors is induction of peripheral vasoconstriction to raise SVR, either through the α_1 adrenergic or V_1 (vasopressin) vascular smooth muscle receptors, ultimately leading to increased preload and CO. Since SVR is 1 of the 2 determinants of MAP, vasopressors are generally titrated to maintain MAP > 65 mmHg. Inotropes all increase myocardial contractility (inotropy) by activating myocardial β_1 adrenergic receptors, directly increasing CO. β_1 receptors also have a chronotropic effect on the sinoatrial and atrioventricular nodes, increasing heart rate. Notably, inotropes also activate β_2 adrenergic receptors on peripheral vascular smooth muscle, leading to peripheral vasodilation, decreased SVR and MAP, and a potential decrease in preload and CO (although not enough to negate the increase achieved with β_1 activation). Therefore, inotropes are titrated to achieve acceptable CO, chamber filling pressures, and markers of end-organ perfusion with an eye to avoiding excessive hypotension that may counteract those goals.

The most commonly used vasopressors are phenylephrine, norepinephrine, and epinephrine (Table 2).[23–25] Phenylephrine acts only on α_1 adrenergic receptors and is therefore considered a pure vasopressor that is commonly used peri-

Table 2 Vasoactive and Inotropic Agents Used in the Management of Shock

Pharmacologic Agent	Target Receptors		Typical Dose	SVR	HR	CO	Adverse Effects
	Cardiac	Vascular					
Norepinephrine	β_1: ++	α_1: +++++ β_2: +	0.02-3 μg/kg/min	↑↑↑	↑	↑/↓	Tachyarrhythmias, bradyarrhythmias, cardiac myocyte apoptosis, peripheral ischemia, HTN (especially in nonselective β-blocked patients)
Epinephrine	β_1: ++++	α_1: ++++ β_2: +++	0.01-0.20 μg/kg/min	↑	↑↑↑	↑↑	Ventricular arrhythmias, severe HTN leading to cerebrovascular events, peripheral ischemia, metabolic acidemia, cardiac ischemia
Phenylephrine	β_1: none	α_1: ++++ β_2: none	Initial: 100-180 μg/min Maintenance: 40-60 μg/min	↑↑↑	↓	↓	Worsening cardiac function, reflex bradycardia, HTN (especially in nonselective β-blocked patients)
Vasopressin	β_1: none	V_1, V_2	0.01-0.04 U/min	↑↑↑	↓	↓	Tachyarrhythmias, digital ischemia
Dopamine	β_1: ++++	α_1: +++ β_2: ++ DA: ++++	2-20 μg/kg/min	↓/↑ (↓ at lower doses, ↑ at higher doses)	↔/↑ (↑ at higher doses)	↑	Tachyarrhythmias†, cardiac ischemia, severe HTN
Dobutamine	β_1: ++++	α_1: + β_2: ++	5-15 μg/kg/min	↓	↑↑↑	↑↑	Ventricular arrhythmias, cardiac ischemia, hypotension
Milrinone	β_1*: ++++	α_1: none β_2*: ++++	0.25-0.75 μg/kg/min	↓↓	↑↑↑	↑↑	Vasodilatory effects mediated through PDE3 inhibition, ventricular arrhythmias

Abbreviations: CO, cardiac output; HR, heart rate; HTN, hypertension; SVR, systemic vascular resistance.

*Milrinone acts via inhibition of PDE3 degradation of cAMP, with effects equivalent to actions on β_1 and β_2 receptors.

†While dopamine has previously been used in septic shock, most clinicians avoid this agent in this population as there is a higher risk of cardiac arrhythmias with no significant benefit over other agents such as norepinephrine or vasopressin.

procedurally to counteract anesthesia-related vasoplegia. Norepinephrine and epinephrine have both α_1 and β_1 adrenergic activity—due to its versatility and overall favorable safety profile, titratable norepinephrine infusion is the first line vasopressor in undifferentiated shock. Epinephrine has stronger β_1 activity than norepinephrine and can be utilized in most types of shock, but particularly as a pseudo-inotrope in cardiogenic shock and right ventricular failure. However, it also carries β_2 activity that counteracts some of the peripheral vasoconstriction from α_1 agonism; therefore, its effect on SVR is not as robust as other vasopressors. Vasopressin causes strong peripheral and splanchnic vasoconstriction through V_1 receptors and is typically used as an adjunctive, fixed-dose agent once high doses of the first-line pressor are reached. Prolonged and high-dosage use of vasopressors can induce digital and mesenteric ischemia.

Dobutamine and milrinone are inotropes used to treat cardiogenic shock (Table 2).[23–25] Both strongly activate β_1 receptors with moderate β_2 receptor activation, leading to desired increased myocardial contractility with the generally undesired effect of decreased SVR. Vasopressors can be utilized alongside these inotropes to counteract this vasodilation. Milrinone, as a phosphodiesterase inhibitor, also causes pulmonary vasodilation, making it very effective in cases of right ventricular failure as both an inotrope and RV afterload reducer. Any agent that activates β_1 receptors (norepinephrine, epinephrine, and all inotropes) will induce chronotropy in addition to inotropy, and tachyarrhythmias can be seen with use — these generally subside upon discontinuation.

Strategies, Monitoring, and Endpoints of Resuscitation

In all instances of shock, the goal of treatment is to restore aerobic metabolism and end-organ function by restoring CO. Objective clinical indicators of organ perfusion that are commonly monitored in shock include urine output, serum lactate, and mixed/central venous oxygen saturation (SvO2/ScvO2). Renal perfusion and oxygen delivery are commonly impaired in shock due to reduced CO, leading to oliguria, commonly defined as urine output less than 0.5 ml/kg/hr.[26,27] Oliguria may improve rapidly if shock is short-lived and treated expeditiously; however, if renal ischemia was present long enough to cause tubular necrosis, glomerular filtration may remain impaired despite recovery of CO and perfusion.

When tissues experience decreased oxygen delivery in shock, they are forced to transition from aerobic to anaerobic metabolism at a certain point. Lactic acid, a byproduct of anaerobic metabolism, accumulates in the serum as a result. Both initial lactate level and rate of clearance are prognostic. Serum lactate level greater than 1.0-2.0 mmol/l is considered elevated.[28] The arterial base deficit obtained on ABG is an indicator of the presence of metabolic acidosis (considered elevated if >2 mmol/l) and can be used as a proxy indicator of lactic acidosis in the correct setting.

Mixed and central venous oxygen saturation (SvO2 and ScvO2) are described in a previous section and are generally tracked in cases of cardiogenic and hypovolemic shock to assess global tissue perfusion status.

Normalization of oxygen delivery in shock typically requires support of CO through reversal of etiology-specific hemodynamic derangements. The cornerstone of management of hypovolemic shock is restoration of preload via IV crystalloid or colloid administration. Vasopressors that increase SVR ± myocardial contractility such as norepinephrine, phenylephrine, and epinephrine may be used as adjuncts to maintain a perfusing blood pressure while intravascular volume is restored. Once patients can maintain a MAP > 65 mmHg without requiring additional volume or vasopressor support, this can be taken as an indicator of adequate CO.

Distributive shock can only be corrected by treating the underlying etiology causing vasoplegia, but counteracting the decrease in SVR with α-agonist vasopressors is usually required to support CO and generate a perfusing pressure in the meantime. Fluids can also be administered to replenish preload, as vasoplegia causes "pooling" of venous blood in the periphery and decreased venous return. As with hypovolemic shock, maintaining MAP > 65 mmHg is a sufficient hemodynamic resuscitation goal.

With cardiogenic shock, particularly left ventricular failure, the end-goal is correction of CI rather than normalization of blood pressure alone. MAP can be normalized with vasopressors or fluids while end-organ perfusion may still be inadequate due to impaired CO, giving a false sense of security. Therefore, continuous tracking of CI/CO using PAC or APWA can be very helpful in management. To improve CO, the targets of treatment are optimization of preload (chamber filling), reduction of afterload, and support of myocardial contractility. Contractility can be assessed qualitatively (ventricular squeeze) and quantitatively (ejection fraction and SV) using FoCUS/echocardiography. Inotropes and vasopressors with β_1 agonist activity are excellent options to increase contractility. When forward flow out of the heart is impaired, the chambers can become overfilled, which can further compromise contractility — pathologically elevated RV and LV preload can be visualized sonographically or measured as elevated CVP and/or PAOP through a PAC, and IV diuresis is indicated. Of course, care must be taken to avoid overdiuresis, which will lead to recurrent depression of CI/CO. One important etiology of cardiogenic shock that is managed differently than most other causes is dynamic left ventricular outflow tract obstruction (LVOTO). LV output is impaired in dynamic LVOTO when pre-existing LV outflow tract narrowing is exacerbated by hypovolemia or increased velocity through the LVOT—in these cases, diuresis and inotropy can be disastrous, and the treatment is IV fluid administration and atrioventricular node blockade with β- or calcium channel-blockers. Diagnosis of LVOTO usually requires echocardiography by an experienced provider.

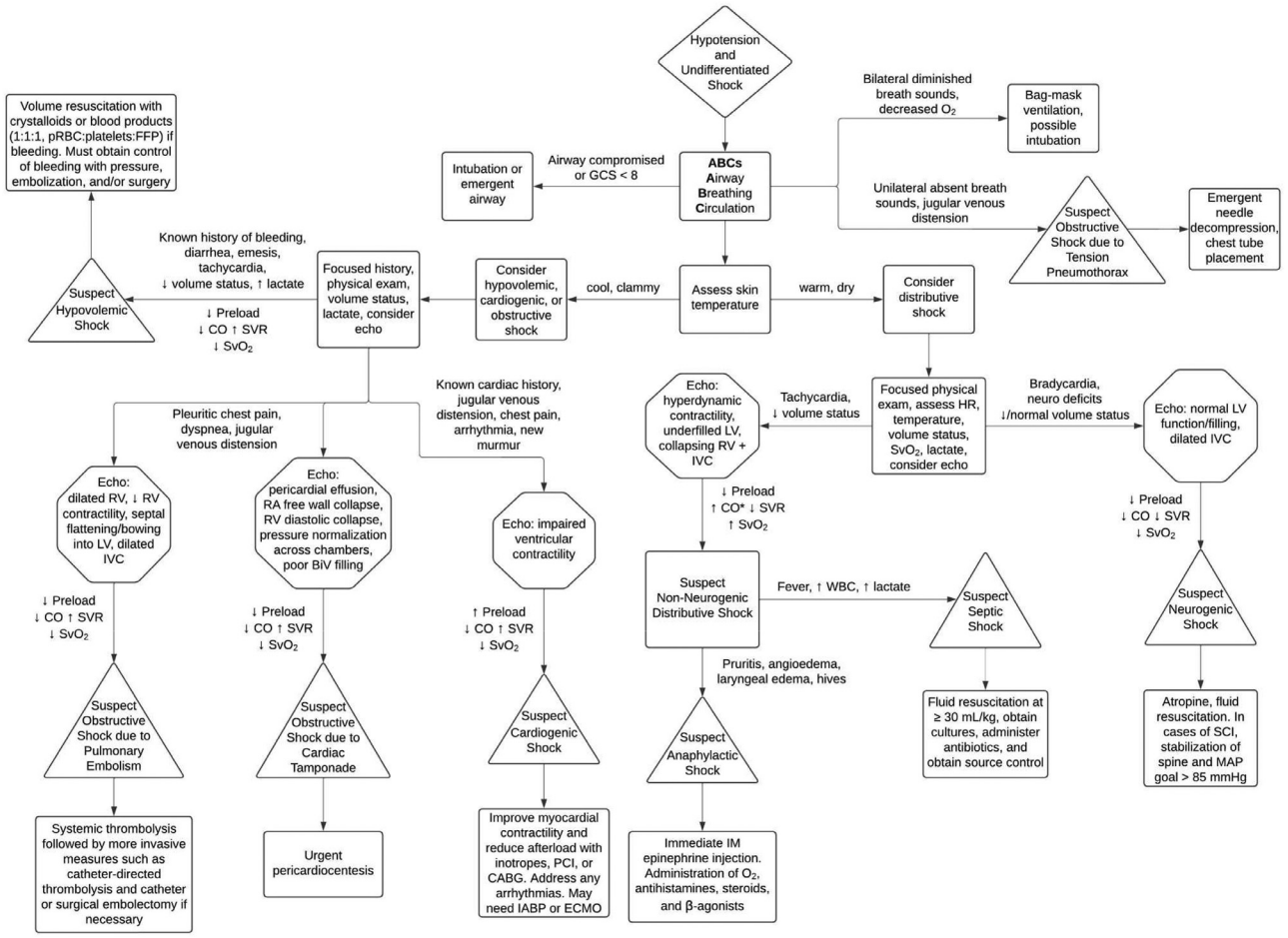
Hypotension and Undifferentiated Shock
ABCs Airway Breathing Circulation
Airway compromised or GCS < 8
Intubation or emergent airway
Bilateral diminished breath sounds, decreased O_2
Bag-mask ventilation, possible intubation
Unilateral absent breath sounds, jugular venous distension
Suspect Obstructive Shock due to Tension Pneumothorax
Emergent needle decompression, chest tube placement
Assess skin temperature
cool, clammy
Consider hypovolemic, cardiogenic, or obstructive shock
warm, dry
Consider distributive shock
Focused history, physical exam, volume status, lactate, consider echo
Known history of bleeding, diarrhea, emesis, tachycardia, ↓ volume status, ↑ lactate
↓ Preload ↓ CO ↑ SVR ↓ SvO_2
Suspect Hypovolemic Shock
Volume resuscitation with crystalloids or blood products (1:1:1, pRBC:platelets:FFP) if bleeding. Must obtain control of bleeding with pressure, embolization, and/or surgery
Pleuritic chest pain, dyspnea, jugular venous distension
Known cardiac history, jugular venous distension, chest pain, arrhythmia, new murmur
Echo: dilated RV, ↓ RV contractility, septal flattening/bowing into LV, dilated IVC
Echo: pericardial effusion, RA free wall collapse, RV diastolic collapse, pressure normalization across chambers, poor BiV filling
Echo: impaired ventricular contractility
↓ Preload ↓ CO ↑ SVR ↓ SvO_2
↓ Preload ↓ CO ↑ SVR ↓ SvO_2
↑ Preload ↓ CO ↑ SVR ↓ SvO_2
Suspect Obstructive Shock due to Pulmonary Embolism
Suspect Obstructive Shock due to Cardiac Tamponade
Suspect Cardiogenic Shock
Systemic thrombolysis followed by more invasive measures such as catheter-directed thrombolysis and catheter or surgical embolectomy if necessary
Urgent pericardiocentesis
Improve myocardial contractility and reduce afterload with inotropes, PCI, or CABG. Address any arrhythmias. May need IABP or ECMO
Focused physical exam, assess HR, temperature, volume status, SvO_2, lactate, consider echo
Tachycardia, ↓ volume status
Bradycardia, neuro deficits ↓/normal volume status
Echo: hyperdynamic contractility, underfilled LV, collapsing RV + IVC
Echo: normal LV function/filling, dilated IVC
↓ Preload ↑ CO* ↓ SVR ↑ SvO_2
↓ Preload ↓ CO ↓ SVR ↓ SvO_2
Suspect Non-Neurogenic Distributive Shock
Fever, ↑ WBC, ↑ lactate
Suspect Septic Shock
Suspect Neurogenic Shock
Pruritis, angioedema, laryngeal edema, hives
Fluid resuscitation at ≥ 30 mL/kg, obtain cultures, administer antibiotics, and obtain source control
Atropine, fluid resuscitation. In cases of SCI, stabilization of spine and MAP goal > 85 mmHg
Suspect Anaphylactic Shock
Immediate IM epinephrine injection. Administration of O_2, antihistamines, steroids, and β-agonists

Figure 1 Algorithmic approach to undifferentiated shock. Abbreviations: CABG, coronary artery bypass graft; CO, cardiac output; ECMO, extracorporeal membrane oxygenation; FFP, fresh frozen plasma; GCS, Glasgow Coma Scale; IABP, intra-aortic balloon pump; IVC, inferior vena cava; LV, left ventricle; PCI, percutaneous coronary intervention; pRBC, packed red blood cells; RV, right ventricle; SCI, spinal cord injury; SvO_2, mixed venous oxygen saturation; SVR, systemic vascular resistance; WBC, white blood cell; *May be decreased in late septic shock.

Ultimately, severe cases of cardiogenic shock may require placement of mechanical circulatory support devices such as intra-aortic balloon pump (IABP), left ventricular assist device (LVAD), or veno-arterial extracorporeal membrane oxygenation (VA-ECMO) in addition to fluid optimization and inotropy to generate CO sufficient for organ perfusion.[29]

Emergent hemodynamic support is crucial in obstructive shock and should be enacted concurrently with echocardiographic/cross-sectional imaging and mobilization of resources to correct the underlying etiology. In cases of obstructive shock due to increased pulmonary vascular resistance such as massive PE or pulmonary hypertension, right ventricular failure can occur due to acutely elevated pressures and volumes. Sonographic evaluation of intravascular fluid status with corresponding IV fluid or diuretic administration, administration of vasopressors and/or inotropes that do not increase pulmonary vascular resistance, and RV afterload reduction with inhaled nitric oxide or epoprostenol should be performed to avoid ensuing RV failure. With cardiac tamponade or tension pneumothorax/pneumoperitoneum, any available hemodynamic support including fluids and vasopressors/inotropes should be given while emergent decompression is prepared and performed.

Conclusion

Prompt recognition and management of undifferentiated shock are paramount to preventing irreversible organ damage and improving patient outcomes. IRs play a critical role in this process, especially when complications arise during procedures or in the acute care of critically ill patients. Management of shock requires a systematic approach that includes immediate stabilization of airway/breathing/circulation, identification and treatment of underlying etiologies, hemodynamic assessment, and hemodynamically guided resuscitation. This comprehensive framework will allow IRs to more confidently manage undifferentiated shock while ensuring a timely transition to definitive care (Fig. 1).

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